

NOTES

TYPE III HYPERSENSITIVITY REACTIONS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Hypersensitivity reaction mediated by immune complexes
 - Antibodies (IgG) binding to soluble antigens → antigens not bound to cell surfaces
- Formation of immune complexes → complement activation (esp. C3a, C4a, C5a)
 - Anaphylatoxins → increase vascular permeability → edema
 - Chemokins → recruitment of phagocytes, neutrophils, mast cells → degranulation of lysosomal enzymes, reactive oxygen species → inflammation, tissue necrosis (fibrinoid necrosis)
 - May also elicit systemic inflammation
- Common sites of immune complex accumulation
 - Blood vessel walls → vasculitis
 - Kidneys → glomerulonephritis
 - Joints → arthritis
- If triggered by single exposure to antigen, resolves after catabolism of immune complexes → acute serum sickness
- If repeated/prolonged exposure → chronic serum sickness
 - Systemic erythematosus lupus (SLE), polyarteritis nodosa, poststreptococcal glomerulonephritis, reactive arthritis

Common Type III hypersensitivity reactions

- Henoch–Schönlein purpura
- Hypersensitivity vasculitis
- Reactive arthritis
- Farmer's lung
- Post-streptococcal glomerulonephritis
- Serum sickness
- Arthus reaction

- Systemic lupus erythematosus
- Subacute bacterial endocarditis
- Rheumatoid arthritis

SIGNS & SYMPTOMS

- Fever, fatigue, weight loss
- **Skin:** rash, urticaria
- **Kidney:** proteinuria
- **Joints:** arthralgias
- **Mucosa:** ulcers
- **Serosa:** pleuritis, pericarditis

DIAGNOSIS

LAB RESULTS

- Antibody testing
- Histopathology to observe fibrinoid necrosis
 - Necrosis of vessel wall with infiltration of neutrophils, eosinophils, complement, plasma proteins; staining pattern reminiscent of fibrin

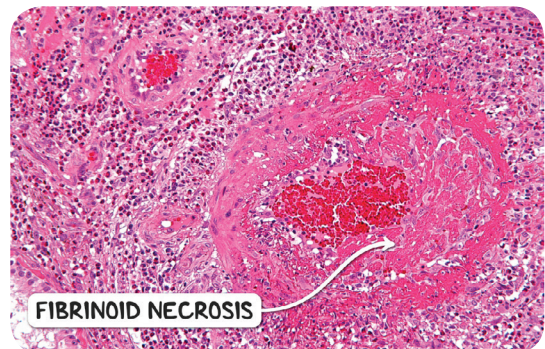


Figure 38.1 A vessel displaying fibrinoid necrosis in Churg–Strauss syndrome, which is a type III hypersensitivity reaction.

VISIT...

LANZAROTE
Caliente.COM

- Immunofluorescence microscopy to visualize immune complexes
- Complement levels in blood
 - Track disease progression

TREATMENT

MEDICATIONS

- SLE
 - Administration of anti-inflammatory, corticosteroids, cytotoxic medications to decrease inflammatory activity

SERUM SICKNESS

osms.it/serum-sickness

PATHOLOGY & CAUSES

- Systemic Type III hypersensitivity reaction **against foreign antibodies in serum**
- Exposure to foreign serum → triggers B cells to become plasma cells, produce IgG antibodies against foreign antibodies → **immune complexes formed, deposited in basement membrane** → complement activation, immune cells recruitment → lysosomal enzymes, reactive oxygen species, cytokines produced → local, systemic inflammatory response
- Initial exposure
 - **4–10 days to develop** reaction
- Second exposure
 - Faster, more potent reaction
- Resolves after withdrawal of culprit agent

CAUSES

- **Medications** (e.g. cefaclor, anti-seizure medications); infections (e.g., hepatitis B); vaccines

SIGNS & SYMPTOMS

- Allergy-like response
- **Present within 1–2 weeks** if initial exposure, 12–36 hours if second exposure
 - General malaise, erythema, itching, **arthralgia**
- **Fever** > 38.5°C/101.3°F, rash, **lymphadenopathy**, polyarthrititis, proteinuria

DIAGNOSIS

LAB RESULTS

- Complete blood count (CBC)
 - Neutropenia, increase in lymphocytes
- Elevation of erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)
- Urinalysis
 - Mild **proteinuria** (50%)

OTHER DIAGNOSTICS

- Clinical presentation; suspected when allergy-like symptoms present after administration of potential responsible agent (e.g. antivenom serum after bitten by snake)
- Testing to exclude infections (e.g. hepatitis B)

TREATMENT

MEDICATIONS

- Relieve symptoms with antihistamines, analgesics
- Glucocorticoids for individuals with severe clinical features (e.g. very high fever, severe arthritis, extensive rashes)

OTHER INTERVENTIONS

- Discontinue, avoid offending agent